AMENDMENTS TO THE SPECIFICATION:

Page 5, please amend the paragraph beginning on line 16 with the following amended paragraph:

--FIG. 4 is the sequence alignment of Avd and the AVRs (AVR1-AVR7) (SEQ ID NOS 3-9 respectively in order of appearance). Dots indicate identical amino acids in AVRs as compared to avidin and the two amino acid deletion in AVRs is indicated by dashes. Horizontal arrows designate the β-sheets of avidin, and the vertical arrow indicates the cleavage site of the signal peptide in avidin. Biotin-binding residues are bold-faced in the avidin sequence, and the N-glycosylation site of avidin as well as the potential N-glycosylation sites of the AVRs are highlighted with gray. Cysteine residues are boxed.--

Page 6, replace the paragraph beginning on line 24 with the following amended paragraph:

--FIG 9. Sequence alignment of chicken avidin (SEQ ID NO: 10) and AVR4/5 (SEQ ID NO: 11). The secondary structure of chicken avidin is also shown (β-strands: arrows). Mutated amino acid residues are shaded with black. Amino acid residues located at the monomer-monomer interfaces are boxed. Potential differences explaining the high thermal stability of AVR4/5, in comparison to avidin, are shaded in gray.--

Page 8, replace the paragraph beginning on line 4 with the following amended paragraph:

--Avidin ~~(SEQ ID NO 1)~~ and streptavidin ~~(SEQ ID NO 2)~~ are valuable and widely used tools in the life sciences. In addition to their high biotin-binding affinity, the robustness and the flexibility of the system relies on their extreme stability under various demanding conditions. In the present application one object is to increase the stability of chicken avidin and other biotin binding proteins even further without losing its strong biotin-binding ability. In order to achieve this goal two, four or six intermonomeric disulphide bridges are introduced to avidin. The same could also be done to avidin related proteins. In addition, the role of the intramonomeric disulphide bridges in the stability of the avidin tetramer was determined, by removing them using site-directed mutagenesis.--

Page 24, replace the paragraph beginning on line 8 that bridges pages 24 and 25 with the following amended paragraph:

--The AVR1-4/5 genes (Keinänen, R. A., Laukkanen, M. L. and Kulomaa, M. S. (1988) J. Steroid Biochem. **30**, 17-21; Keinänen, R. A., Wallen, M. J., Kristo, P. A., Laukkanen, M. O., Toimela, T. A., Helenius, M. A. and Kulomaa, M. S. (1994) Eur. J. Biochem. **220**, 615-21) were cloned into the EcoRI-HindIII sites of pGEM4 and were in vitro transcribed and spliced using Ribomax and RNA Splicing System kits (Promega) according to the manufacturer´s instructions. The cDNAs were then produced by RT-

PCR and further amplified by PCR using the oligonucleotide primers: AK33 (5'-CTGCTAGATCTATGGTGCACGCAACCTCCCC-3') (SEQ ID NO: 1) and AK44 (5'-GTTGCAAGCTTTGCGGGGCCATCCT-3') (SEQ ID NO: 2) containing BglII and HindII restriction sites, respectively. After cutting with BglII and HindIII, the AVR1-4/5 cDNAs were cloned into BamHI and HindIII sites of pFASTBAC. For AVR6 and AVR7, cDNAs were produced by subcloning the corresponding genes (Ahlroth, M. K., Kola, E. H., Ewald, D., Masabanda, J., Sazanov, A., Fries, R. and Kulomaa, M. S. (2000) Anim. Genet. **31**, 367-75) into pDsRed1 (Clontech) where the red fluorescent protein-encoding region had been removed, and by subsequent transfection of the constructs into NIH/3T3 cells. Total RNA was extracted from the cells using the SV Total RNA Isolation System (Promega). The AVR6 and AVR7 cDNAs were produced by RT-PCR (RobusT RT-PCR Kit, Finnzymes, Espoo, Finland) using the oligonucleotide primers AK33 and AK44 to produce BglII and HindIII restriction sites to the 5'- and 3'-ends of the cDNAs, respectively. The synthesised cDNAs were cloned into BamHI/HindIII digested pFASTBAC1, the cloning vector for the Bac-To-Bac Baculovirus Expression System (Gibco BRL, Life Technologies, Gaithersburg, MD, USA). The nucleotide sequences of the cDNAs were confirmed by sequencing. The virus vectors for producing the AVR proteins were constructed and amplified according to the Bac-To-Bac system instructions. Recombinant AVR proteins were produced in Sf9 insect cells as previously reported (Airenne, K. J., Oker-Blom, C., Marjomäki, V.

S., Bayer, E. A., Wilchek, M. and Kulomaa, M. S. (1997) Protein Expr. Purif. **9**, 100-10822). Proteins were purified from the cells using affinity chromatography on a 2-iminobiotin (AVR1, AVR3, AVR4/5-7) and/or biotin agarose column (AVR1 and AVR2) as previously described (Laitinen, O. H., Airenne, K. J., Marttila, A. T., Kulik, T., Porkka, E., Bayer, E. A., Wilchek, M. and Kulomaa, M. S. (1999) FEBS Lett. **461**, 52-8). AVR2 was eluted from biotin agarose with 1 M acetic acid and the elution fractions were immediately neutralised with NaOH. The elution of AVR1 was achieved using 1 M HCl followed by neutralisation with Tris (1 g/ml).--